UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF OHIO
WESTERN DIVISION

LUANN PARKER,

Plaintiff,

vs. CIVIL ACTION NO. C-1-00-766

AVENTIS S.A., et al.,

Defendants.

DEPOSITION OF: DAVID A. GRIESEMER

DATE: July 15, 2003

TIME: 9:19 a.m.

LOCATION: Hampton Inn
1104 Isle of Palms Connector
Mount Pleasant, SC

TAKEN BY: Counsel for the Defendants

REPORTED BY: LISA F. WALKABOUT,
Court Reporter

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STIPULATION

It is stipulated by and between Counsel that this deposition is being taken in accordance with the Federal Rules of Civil Procedure; that all objections as to Notice of this deposition are hereby waived; that all objections except as to form are reserved until the time of trial; and that the witness does not waive reading and signing of this deposition.

* * * * * * * * * * *

DAVID A. GRIESEMER

being first duly sworn, testified as follows:

MR. SICILIANO: For the record, we are here today for the deposition of David A. Griesemer, M.D. taken in this case pursuant to agreement of the parties; is that correct?

MR. NAMEI: Yes.

EXAMINATION

BY MR. SICILIANO:

Q. And am I pronouncing your name right, Griesemer?

A. Griesemer.

Q. Griesemer. Why don't you introduce yourself and state your name for the record.

A. My name is David Griesemer.

Q. And where do you live?

A. In Mount Pleasant, South Carolina.

Q. What is your business address?

A. Department of Neurology, Medical University of South Carolina, 96 Jonathan Lucas Street, Charleston, South Carolina, 29425.

Q. What are your present duties as a doctor at the medical college of South Carolina?

A. I work as a clinician at the Medical University seeing patients for a portion of my time. I'm al -- I'm also chairman of the Department of Neurology, so I have administrative and teaching responsibilities associated with that.

Q. What courses do you teach presently?

A. I participate in teaching of the first-year medical neurosciences course. I also participate in teaching in the third-year core neurology rotation. I'm also responsible for teaching neurology residents who have already earned their MD degree, and, under special circumstances, I'll also be responsible for teaching pediatric residents, psychiatry residents or other subspecialists rotating through the

neurology service.

Q. Do you have any teaching roles where

you are the primary professor for a particular class?

A. No.

Q. Have you ever had that?

A. No, that's not typically the case for clinicians who usually work one-on-one alongside a resident or student in training at the bedside.

Q. You say you also have a role as a clinician.

A. Yes.

Q. Tell me what that means. What do you do?

A. What it means is that, one day each week, I'm in an outpatient setting seeing patients. It also means that I take my equitable share of supervising the residents in their continuity clinic as they see their patients. It means that, for three to four months out of the year, I'm on the inpatient consult attending service, you know, responding to requests for consultation by other physicians at the university hospital.

Q. What other duties do you have?

MR. NAMEI: Isn't that enough?
THE WITNESS: Those that I've mentioned really occupy most of my time.

BY MR. SICILIANO:

Q. Most of your time? Okay. We're here today because you're acting as an expert on behalf of the Plaintiff in this case. Can you tell me how often do you do that, that is, provide medical/legal consultation?

A. I provide medical/legal consultation probably about a dozen times a year in terms of reviewing records for either the plaintiff or defense or prosecution or the defense, the claimant or the respondent. Perhaps half of those may go to deposition, and a small percentage of those may go to trial.

Q. Can you give me a sort of a percentage of how many of those medical/legal cases you were involved in -- you have been involved in involved civil litigation versus criminal litigation?

A. I would say that probably 40 percent of the cases are malpractice-related, 40 percent are other civil matters and 20 percent are criminal.

Q. Of the other civil matters, what kind

of cases do you handle?

A. These would be patients who may have sustained neurologic injury through -- through trauma, exposure to a product, some other

misadventure.

Q. Can you tell me, of the cases that you have handled in the civil arena, how many of them have been for the defense?

A. I don't recall specifically, but I would say they're probably evenly divided for plaintiff and defense with the possible exception of some involvement I've had with litigation related to lead toxicity in children and that's more heavily weighted toward the plaintiff.

Q. Have you been involved in a number of lead toxicity cases?

A. Yeah, probably half a dozen over the years.

Q. Going over the past five years, can you tell me, in those cases that you have been consulting on behalf of plaintiffs or defendants, what products, if products were involved, were involved in those kinds of litigations?

A. The two that come to mind, one product

was an ephedra compound produced by Rexall. The other was a -- was Prozac marketed by Lilly.

Q. Well, let's talk about the ephedra compound. What did that case or those cases involve?

A. This was a circumstance in which a woman expired after -- after her family claimed she had had exposure to ephedra. And, you know, there were a variety of compounding circumstances in the case.

Q. And you testified on behalf of Rexall or the --

A. On behalf of the defense, yes.

Q. How did neurology play a role in that case?

A. Because the event was a stroke, and, while most of the literature relates to cardiac complications, the issue was whether this was related.

Q. Have you ever handled an or consulted on any ephedra case for the plaintiff?

A. No.

Q. Let's talk about the Prozac. Did you consult on behalf of the plaintiff or the defendant

in the Prozac litigation?

A. On the defense.

Q. That was for Eli Lilly?

A. Yes.

Q. What did those cases involve?

A. This was the case of a boy with

Tourette's syndrome who had committed suicide in the context of being placed on Prozac.

Q. And I take it your opinion was the Prozac did not cause the individual to commit suicide?

A. That's correct.

Q. Approximately how many cases have you handled on behalf of Eli Lilly?

A. As far as I know, that was the only one.

Q. And the same question with regard to Rexall --

A. The only --

Q. -- just one?

A. Yes.

Q. Have you ever testified or consulted on any vaccine cases?

A. Yes.

Q. Which cases were those and what vaccines were involved?

A. I don't have a clear or accurate recollection. There were a series of perhaps a half dozen cases in the early 1990s that were brought to a hearing under the National Vaccine Injury Act, and my recollection is these involved

DPT and MMR vaccinations.

Q. And were you engaged by the petitioners or the plaintiffs in that case?

A. Probably 80 percent of the time, I represented the petitioner and about 20 percent of the time representing the respondent.

Q. In general, what was your opinion on those kinds of cases?

A. I don't know that I had a general opinion, it depended on the specifics of the case. Some of them I felt were meritorious claims, and some of them I felt were not.

Q. Do you remember -- let's just talk about the DPT cases -- what kind of injuries the child had that you claimed would have been --

A. No, I'm sorry.

Q. -- related to the DPT?

A. I don't recall. My long-term memory is not that good, and I didn't review those in preparation for today's deposition.

Q. Approximately how many depositions have you given over the last ten years?

A. I don't know exactly. I would estimate maybe 40 to 50, perhaps four or five a year. That could be high, I'm not sure.

Q. Have you ever consulted with either defense or plaintiffs in connection with an influenza vaccine?

A. Not that I recall.

Q. So this is the first, to the best of your knowledge?

A. I believe so, yes.

Q. Have you ever administered influenza vaccine?

A. Not personally, no.

Q. Have you ever prescribed it?

A. I've recommended it, and I've received it.

Q. In what context have you recommended it?

A. I usually recommend it to higher-risk

patients who may be particularly vulnerable to the effects of -- of getting influenza. The patients I follow regularly tend to be patients with epilepsy and that may be a particularly vulnerable population. So typically, my more fragile patients, I'll recommend they consider a vaccine.

Q. Do you have any opinions about the effectiveness of influenza vaccine?

A. I don't have any opinions apart from

what's available in the general medical literature. I think that, on the whole, I find them beneficial.

Q. Why did you take influenza vaccine?

A. Because I don't like getting the flu.

Q. Has any of your -- have any of your family members received the influenza vaccine?

A. Not that I'm aware of. I'm the one who's primarily in a patient-related environment, more at risk because of my job.

MR. SICILIANO: Let's mark this as an exhibit.

(DFT. EXH. 1, Curriculum Vitae, was marked for identification.)

(Off-the-record conference.)

BY MR. SICILIANO:

Q. Doctor, I'm going to hand you what has been marked as Defendant's Exhibit Griesemer 1 and ask you to identify that for the record, please. (Tendering)

A. It looks like a copy of my current CV.

Q. And I'm just going to go over parts of it with you. Why don't you just briefly tell us about your educational history and then your professional history after med school.

A. All right. I received my undergraduate

degree in human biology from Johns Hopkins in 1973. I received my MD from Hopkins in 1976. I remained there for two years as an intern and resident in pediatrics.

At that point, I interrupted my formal studies and took a position with the U.S. Public Health Service. I spent four years in Northern Arizona on the Navaho and Hopi Indian Reservation as a general medical officer, delivered babies, set fractures, taking care of myocardial infarctions.

After four years, I decided to continue my training, and I went to the University of Michigan where I did three years of training in neurology, making me board eligible in neurology

with special competence in child neurology.

From there, I returned to Northern Arizona and established a private practice in Prescott where I worked for four years. After that experience, I moved to Tucson and, in Tucson, took a position on the faculty of the University of Arizona. So that was the beginning of my academic career, and I believe that was in 1990.

In 1993, I moved from the University of Arizona to the Medical University of South Carolina, and I've been here since then, starting

as an assistant professor, moving up to associate professor, then associate professor with tenure and then a full professor. I became chairman of the department in 2000.

Q. Looking at your CV, it appears that you have a specialty in -- is it pediatric neurology?

A. Yes.

Q. Are the patients that you treat in the clinics that you run or participate in more focused on children?

A. Yes, they are.

Q. Do you treat adults?

A. Yes.

Q. How often do you treat adults?
A. It -- it depends on the circumstances. Sometimes I will follow adults with epilepsy in my clinics. When I'm supervising residents in their continuity clinic, I'm responsible for evaluating and treating adult patients. When I do outreach into the developmental centers throughout the state, those are patients of all ages, but, because of their profound handicap, are most comfortably cared for by pediatric neurologists.
Typically, adult neurologists like to be able to talk to their patients, and so the

pediatric neurologist oftentimes has some special skills in dealing with noncommunicative patients. So the majority of my patients are pediatric neurology patients, but I certainly teach and supervise the care of adult patients as well.
Q. You have, according to your CV, written articles, given speeches and done -- and done some publications in the electronic media. Have you ever written any article concerning acute disseminated encephalomyelitis?
A. No.
Q. Have you ever studied -- and I'm just

going to refer to this as -- ADEM?

A. In terms of a scientific study or a patient-related study, no.

Q. What's your familiarity with ADEM in your practice?

A. My familiarity with it is as a disorder that I diagnose and see in my patients from time to time. ADEM is something that is more commonly seen in children than in adults, so pediatric neurologists tend to have perhaps a greater familiarity with it than adult neurologists have.

I would say I have no particular expertise concerning ADEM that exceeds that of any

good neurologist with a broad exposure to clinical disorders.

Q. Are you associated with any expert groups at all?

A. I have allowed my name to be listed with Park Dietz Associates.

Q. Spell that for me.

A. Park is P-A-R-K, Dietz is D-I-E-T-Z, Associates. Dr. Dietz is a psychiatrist who was a friend of mine in medical school, and he has assembled a consultative team of people involved in

primarily criminal forensic matters. And while his group is primarily weighted towards psychiatrists and psychologists, I agree, from time to time, to serve as a neurologic consultant for that group.

Q. Where are they located?

A. California.

Q. How often do you receive referrals from the Park Dietz Associates?

A. Probably once a year. I tend to decline more referrals than I accept simply because of other demands on my time.

Q. And you indicated that that's primarily criminal?

A. Yes.

Q. Defense related?

A. In the two cases I'm thinking of, I've been retained by the prosecution or the U. S. Attorney's office.

Q. I see that you have some publications on your CV that are listed as peer review journals and some that are non-peer reviewed publications. Could you explain the differences there?

A. Peer reviewed articles tend to be original scientific works that are submitted to a

journal and sent out for review by experts in the field, and, based upon the reviewers' comments, the editors decide whether or not to accept or deny publication of the journal article.

Non-peer reviewed articles can be of varied nature, but, for the most part, they're articles that are prepared at the request of an editor, and the review is done by the editor himself or herself before they're brought into publication. Typically, those are review articles or summary articles rather than original research.

Q. And I think you've already testified to this, but let's make sure that none of the articles and none of the publications that you've participated in or written involve ADEM?

A. I believe that's correct.

Q. Tell me how you got involved in consulting on this particular case involving Luann Parker.

A. I received a call from Mr. Namei and that was followed with a letter of February 15th, 2002.

MR. NAMEI: I think what I did was sent him the Complaint, which is, you know, pretty

detailed about the medical history.

THE WITNESS: Right, I have a fax from him dated February 14th with the Complaint. And the following day, apparently he sent me these medical records. (Indicating)

MR. NAMEI: I have no problem if you want to see that, those things. There is nothing confidential in it.

MR. SICILIANO: Let's take a moment just to take a quick look.

THE WITNESS: Okay. (Tendering)

MR. SICILIANO: I might mark a couple of these, this one here. (Tendering)

MR. NAMEI: That's a work product; you can't mark that.

MR. SICILIANO: You don't want me to

look at that? Well, if he relied on it, I get to ask him about it.

MR. NAMEI: Okay, that's fine. I don't see this as any problem. Here, you want a copy? You want to mark it? Go ahead.

MR. SICILIANO: I'm just going to go ahead and mark it, and then what we'll do is, maybe after the deposition, you can send me a copy of

this or maybe we'll leave it with you.

MR. NAMEI: Yes, we can attach it as exhibit 2.

MR. SICILIANO: That's fine.

(DFT. EXH. 2, Typewritten Notes, was marked for identification.)

(Off-the-record conference.)

BY MR. SICILIANO:

Q. Doctor, I'm going to hand you what has been marked as Defendant's Exhibit 2, and please identify that for the record. (Tendering)

A. These are notes that I prepared for myself summarizing the medical records after I initially reviewed them.

Q. Now, did you prepare those or did Mr. Namei or someone in his office?

A. No, these are my notes prepared on my

laptop.

Q. After reviewing medical records?

A. Yes, after reviewing these three volumes of medical records. (Indicating)

Q. And for the record, the medical records that you reviewed are contained in volumes one, two and three, which are notebooks entitled Luann

Parker, Petitioner versus Secretary of Health and Human Services, Respondent?

MR. NAMEI: Yes.

BY MR. SICILIANO:

Q. Is that correct, Doctor?

A. Yes.

Q. And there are three volumes of medical records; is that correct?

A. Yes.

Q. And those medical records were sent to you by Mr. Namei's office?

A. Correct.

Q. And did you review these medical records?

A. Yes.

Q. And from those medical records, you prepared a summary, which is Plaintiff's -- or Defendant's Exhibit 2?

A. I wouldn't pretend that this is a comprehensive summary. These are just my notes to jog my memory about the records.

Q. Did you ever examine Luann Parker?

A. No.

Q. Have you ever spoken to her?

A. No.
Q. Did you ever review any original diagnostic testing?
A. No.
Q. So it's correct to say that you didn't review the original MRIs or CT scans or any of the other diagnostic tests?
A. Not to date, no.
Q. Why don't you tell me, what is acute disseminated encephalomyelitis, which we've been calling ADEM?
A. It's a disorder that involves demyelination in regions of the brain. It is a disorder that is typically monophasic, meaning it's got one phase to it, in distinction to a disorder like multiple sclerosis that also involves demyelination of the brain but has multiple recurrent phases to it.
ADEM is typically a disorder that

follows a viral infection or a vaccination. It begins days to perhaps a week after the vaccination or near the end of the viral infection. Because different parts of the brain can be involved, the clinical manifestations of the disorder are quite

varied.

Oftentimes, there is an involvement of balance or coordination. There may be involvement of alertness, coherence in thinking. There may be focal neurologic signs such as numbness or weakness. In a small percentage of cases, the involvement may include the spinal cord, so there may be disorders of extremity function or bladder function related to spinal cord involvement.

It's a disorder that is diagnosed on the basis of a clinical picture consistent with the pathophysiology. It's also a diagnosis that is usually not made until other items that may resemble it have been satisfactorily ruled out. Those other items may include an infectious encephalitis, may include vasculitis, may include multiple sclerosis, may include multiple strokes.

So typically, the diagnosis is one that is arrived at after a fairly comprehensive look at the patient and after a thoughtful reflection on

the patient's clinical course.

Q. And you have, in the past, diagnosed ADEM --

A. Yes.

Q. -- in your patients? And you said that it's mostly seen in children.

A. More often in children than in adults.

Q. Why is that?

A. Probably relates to the fact that children may be more susceptible to viral illnesses and get them more frequently. It's certainly the case that they're exposed to more vaccinations than adults.

Q. What kind of viral illnesses cause ADEM?

A. There's a wide list of viral types that can cause it. The response is thought to be an autoimmune response where the body responds to an infection, sometimes mistaking its own tissues for the virus to be destroyed, so it's not specific to a particular virus type.

Q. Let me go back to what you have reviewed prior to preparing your opinion in this case. You've indicated you reviewed the three volumes of Mrs. Parker's medical records?

A. Yes.

Q. Did you review anything else?

A. No, not specifically. I did bring with

me two references that are -- are current authoritative sources that look at both the clinical aspect of ADEM and the neuroradiologic aspect of ADEM, but I've not reviewed any specific scientific studies or other literature.

Q. Now, you indicated that -- and maybe I'm interpreting this, but tell me if I'm correct here -- that it's -- this diagnosis is essentially one of exclusion, you rule out certain other diagnoses before you get to ADEM; is that --

A. Well, I agree and disagree with that statement. It is true that, to responsibly make that diagnosis, you need to rule out things that mimic it and that is the process of going through differential diagnosis, but it's also true that there needs to be a certain evolution of events, a certain clinical appearance, certain timing of events that is -- is consistent with ADEM.

What it lacks is a single laboratory test that is confirmatory that makes it possible for us to easily diagnose it with certainty and to the exclusion of other disorders.

Q. So tell me, how do you -- how would you go about diagnosing ADEM in a patient?

A. Well, the first issue is to look at the clinical course of the patient, what has been the spectrum and timing of symptoms that unfold. While some cases of ADEM may have just a very particular symptom and that one symptom only, most cases tend to involve a variety of symptoms.

For example, in Mrs. Parker's case, there were symptoms of unsteadiness or ataxia, symptoms of confusion, symptoms of apraxia, being unable to do common tasks with which she'd previously been familiar, symptoms of sensory impairment, sensories of weakness -- symptoms of weakness.

The multifocal or multifaceted nature of the symptoms suggests first that there's a process that involves more than one particular area of the brain, so that tends to suggest a disorder like ADEM as opposed to a disorder like stroke that may involve just one particular area.

Once characteristic clinical profile is observed, there is a search for appropriate clinical antecedents, had there been a previous viral illness, had there been a previous exposure

to a vaccine, which occurs in the majority of the

cases but doesn't occur in all cases.

And then the final step is to exclude those disorders that I've previously mentioned that could masquerade as ADEM.

Q. Now, how do you go about excluding the infectious encephalopathy --

A. Well --

Q. -- or infectious encephalitis?

A. Right. The easiest way to address that is by looking at spinal fluid through a spinal tap to determine whether there are any inflammatory cells or abnormal chemistries in the cerebral spinal fluid that may indicate infection. That was done in Mrs. Parker's case, and there was no clear evidence of inflammation or infection based upon the spinal fluid.

Looking at other disorders like multiple sclerosis, her physicians looked at other factors in the spinal fluid looking specifically for something called oligoclonal bands that are abnormal proteins often seen in the spinal fluid with multiple sclerosis and that was not seen.

Another item I believe I mentioned was vasculitis or an inflammation of blood vessels, so

there are ways to screen for that, first looking for things like an elevated sedimentation rate that might suggest an inflammatory process. Mrs. Parker did not have that.

I believe, two months after her initial presentation, her physicians at the Cleveland Clinic proceeded to do an angiogram, looking specifically for evidence of an inflammatory vasculitis, and they did not find that.

I talked a little bit earlier about multiple sclerosis which, by definition, involves not only multiple regions of the brain but is a disorder that occurs at multiple points in time. And so the clinical course, Mrs. Parker revealed nothing similar to this before and, to the best of my knowledge, has received -- revealed nothing similar to that subsequently.

The records that I have suggest that she had an illness that progressed to a point of considerable severity and then gradually improved, giving us the temporal characteristics of a monophasic illness consistent with ADEM.

Q. For folks that have ADEM, do they normally recover?

A. The recovery is often good but also

often incomplete. In my experience, just estimating, I would say that patients recover 80 to 90 percent of their previous ability. And these may be motor abilities, they may be cognitive abilities.

Q. And have you examined anything in the recent medical records of Ms. Parker that gives you any idea of the kind of recovery she has undergone?

A. I don't feel I have current records at the moment that would let me answer that question well.

Q. Have you ever treated any patient where you believed the ADEM was caused by some kind of vaccine?

A. Yes.

Q. What kind of vaccines?

A. I think, in the pediatric population, it would be DPT or MMR vaccines.

Q. Have you ever treated any patient who you believed developed ADEM as a result of receiving an influenza vaccine?

A. I don't believe so. I would find that a relatively uncommon occurrence.

Q. Why do you say that?

A. Well, first because I haven't seen it,

and second, it doesn't happen commonly enough that it dissuades people from using the vaccine.

Q. Are you aware of any epidemiological study that has linked influenza vaccine and ADEM in a causal relationship?

A. No, but an epidemiological study that would establish causation for an infrequent occurrence would require an extraordinarily large number of patients to be extensive enough to identify the effect.

Q. You're not an epidemiologist?

A. No.

Q. I'm trying to understand on what basis you have come up with an opinion that Ms. Parker's ADM -- ADEM was caused by an influenza vaccine, so that -- let me walk you through that. You've never treated anyone with ADEM where you have diagnosed it following an influenza vaccine?

A. That's correct.

Q. And you have -- have you?

MR. NAMEI: Excuse me, I don't want to object, but are you assuming that the ADEM that is caused by the influenza vaccine would be different from ADEM that may be caused by, you know, some other problem?

MR. SICILIANO: No, no, I --

MR. NAMEI: Oh, okay.

MR. SICILIANO: -- I'm just asking -- I'm trying to understand the basis for Dr. Griesemer's opinion --

MR. NAMEI: Okay.

MR. SICILIANO: -- so I want him to walk me through this.

BY MR. SICILIANO:

Q. You're not aware of any epidemiological study in peer reviewed medical literature that has ever indicated that there's a cause and effect relationship between influenza vaccine and ADEM?

A. That's correct.

Q. What is the basis for your opinion in this case that Ms. Parker developed ADEM following the influenza vaccine?

A. Well, first, we have evidence that she received an influenza vaccine. Second, we have evidence of symptoms heralding ADEM that begin two days later. Third, we have a characteristic clinical course of ADEM. Fourth, we have clinical experience that says, in most cases, ADEM follows viral illness or vaccination. And finally, we have no evidence of alternative viral illness or

alternative vaccination in temporal proximity to her symptoms.

Q. Are there etiopathic causes for ADEM?

A. There are some cases in which the cause is not easily identifiable, yes.

Q. And have you treated either adults or children with ADEM when you did not know what caused the ADEM in that patient?

A. I have. So the question at hand is, given the fact that she had an immunization and the fact that she had ADEM, I can draw one of two conclusions: I can conclude that there is no relationship whatsoever between these two events, or I can conclude that there is a relationship between these two events. And based upon the preponderance of medical literature and my experience, I believe that it's more likely than not that they are related.

Q. Okay, I want to go and explore the preponderance of medical literature.

A. All right.

Q. Tell me what there -- what exists in the medical literature that leads you to the conclusion or the opinion in this case that the influenza vaccine that she received caused her to

develop ADEM.

A. As I indicated earlier, I have not reviewed any of the early scientific studies concerning ADEM. What I'm reflecting is the current standard of clinical practice. Any --

Q. Tell me, what's that? I don't know what that is.

A. What that means is, in the teaching about ADEM, it is taught to neurologists and neuroradiologists that immunizations can be a cause for ADEM and that influenza immunization is among those causes.

Q. I understand that's what's being taught. I'm trying to explore what's the basis for that.

A. Most likely clinical experience.

Q. All right. Would you agree that, in order to prove a cause and effect relationship between any antigen and a disease, that you need some epidemiological proof that supports that relationship?

A. Well, there are different types of proof. There is scientific evidence that comes from pathological specimens, tissue that's been

biopsied. There is epidemiologic evidence that

makes it statistically probable that there's a cause and effect relationship.

While both of those types of evidence push us to a 95 to 97 percent certainty of cause and effect relationship, neither of those are presently available in this case. There is a different standard for scientific proof than there is for clinical proof than there is for medical/legal proof, and to confuse the standards is a little disingenuous.

You know, clinically, we make a cause and effect relationship where it seems probable and clinically appropriate. That's what the physicians at the Cleveland Clinic did in caring for Mrs. Parker. In reviewing the records and their notes, I concur that that's an appropriate conclusion to draw.

Now, I admit that that does not adhere to the level of scientific proof that I would submit to a peer reviewed journal for publication, but I also assert that it exceeds the level of proof necessary in a medical/legal setting.

Q. Why do you say that?

A. Because I think that the medical/legal standard is more likely than not, and I think the

clinical standard that we've seen in this particular case is very probably the case.

Q. Do you have any appreciation for how many influenza vaccines over the last 25 years have been administered to both children and adults? In the United States, let's state.

A. I would assume that it's an extraordinary number.

Q. And are you aware that, at least since 1976, that governmental agencies like the CDC have engaged in surveillance of adverse effects from or allegedly from influenza vaccines?

A. And I would assume that to be the case.

Q. And with all of those assumptions, you're not aware that any -- there has been any medical epidemiological evidence that influenza vaccine, which has been studied for a long time and given to millions and millions of people, has been associated in a cause and effect relationship with ADEM?

A. I'm not aware that any of that has been published; however, that fact does not change the

observation that Mrs. Parker received a vaccine, nor does it change the observation that she had an episode of ADEM.

Q. Is it fair to say that the basis for your opinion in this case that the influenza vaccine that she received caused her ADEM is the temporal relationship between her receiving the vaccine and her development, in your opinion, of symptoms of ADEM?

A. I believe that's an important factor. I think the other factor is the knowledge and understanding that ADEM, as a clinical disease, is typically triggered by a viral illness or a vaccination. So there's more than just a temporal relationship or coincidence; there is in fact the need for there to be some inciting event. And, in the absence of alternative triggers, the vaccination seems the most likely inciting event.

Q. How, in your opinion, does influenza vaccine trigger ADEM?

A. I don't have the knowledge of an immunologist, but my understanding is that a vaccine or a component of the vaccine may trigger perturbations in T cell function, perhaps

suppressing the subpopulation of T lymphocytes that quiet or control the immune response.

And when those T cells are suppressed, the more aggressive T cells that remain can

initiate the immunological response. I would have to defer to an immunologist who works on a daily basis with what the triggers are for the immunological reaction.

Q. Do all vaccines act alike?

A. In what regard?

Q. In the kind of reactions that a human would have. For example, we've already agreed that there are -- there are no epidemiological studies linking influenza vaccine to ADEM. I assume that you believe that there are -- there is evidence in the literature that other vaccines may cause ADEM.

A. I'm not aware that there's epidemiologic evidence in the literature. Epidemiology is a very useful tool that links causative factors with outcomes, but in order for it to have sufficient power to demonstrate a response, there have to be a sufficient number of cases surveyed. I am not an epidemiologist, but epidemiologic studies are just one of several

approaches to establishing cause and effect relationships.

MR. NAMEI: Can we take a break?

MR. SICILIANO: Sure.

(A recess transpired.)

(DFT. EXH. 3, Affidavit, was marked for identification.)

BY MR. SICILIANO:

Q. Doctor, I'm handing you what has been marked as Defendant's Exhibit 3. (Tendering) Can you identify that for the record?

A. This appears to be my Affidavit with opinions concerning this case.

Q. I want to go over this with you. How was this prepared?

A. I believe the initial draft was prepared by Mr. Namei's office in response to feedback I had given him after reviewing the records. I had the opportunity to review it. I believe there were some minor changes made before I signed it.

Q. I want to go over some portions of Defendant's Exhibit 3 with you --

A. Certainly.

Q. -- beginning with number --
MR. NAMEI: Do you have an extra copy?
MR. SICILIANO: Sure.
MR. NAMEI: Thanks.
MR. SICILIANO: I'm sorry. (Tendering)
BY MR. SICILIANO:

Q. Numbered paragraph 5, 5b, it says, Ms. Parker was free of neurological illness or impairment prior to October 12th, 1998. What's the basis for that statement?
A. Based first on past medical history at the time she was admitted to the hospital, based second upon her level of function as a teacher prior to developing this illness. I think those are primarily the two sources.
Q. Did you review any medical records of her medical history prior to October of 1998?
A. Yes, I believe I did have medical records. There's -- in the medical records, she has a history of high blood pressure dating back to 1986, a history of diabetes dating back to 1993 -- did I say 1986?
THE COURT REPORTER: Yes.
THE WITNESS: Okay. She had been

treated for migraine headaches, and she had problems with increased weight. So there was clear documentation of problems in her medical history, but there was no documentation of neurological problems.

BY MR. SICILIANO:

Q. Can diabetes lead to neurological

problems?

A. It can, yes.

Q. Are migraine headaches some evidence of a neurological problem?

A. Migraine headaches are evidence of neurologic dysfunction. It wouldn't typically cause the kind of impairment that she had. And we did have record of a normal CT scan done back in 1993 as part of a workup for her migraines.

Q. The other subparts contained in paragraph 5, did you obtain those facts from your analysis of the medical records?

A. I believe most of these are derived from either my notes or points that were made in the initial Complaint that are documented in the medical record.

Q. Of these, what would you say are the

most important facts that lead you to the diagnosis in this case of ADEM in Ms. Parker?

A. Well, one important fact is the progressive evolution of symptoms over time. Another important fact is the variety of symptomatology that complicates involvement of different parts of the central nervous system rather than one part.

Q. What does that mean? Tell me what that means.

A. Well, for example, she had difficulty with balance and her walking. That would involve a different part of the nervous system than the region causing her sense of numbness or her left arm weakness. It's important, in looking at her case and in making a diagnosis of ADEM, that she has a diffuse or multifocal involvement of the brain. That makes it less likely that we're dealing with something localized to one region such as a brain tumor or a stroke.

The multifocal nature of the problem is consistent with ADEM. That's borne out, too, with some of the neuroimaging studies that demonstrate a variety of irregularities or abnormalities

involving different areas.

Q. What would you typically expect to see in an MRI or other imaging studies in patients who are suffering from ADEM?

A. Typically, one sees multiple areas of abnormal signal in the white matter region. It tends to occur in regions right around draining veins in the brain. It's somewhat different than the pattern in multiple sclerosis where the white

matter abnormalities are right adjacent to the ventricles or the spinal fluid flow regions.

The abnormalities appear to be of a couple types. There are abnormalities that are indicative of demyelination, and there are abnormalities indicative of breakdown of blood-brain barrier. And you may see both types of abnormalities, the -- the latter suggested by the presence of enhancement when gadolinium is given.

Not all cases of ADEM, however, have evidence of abnormal neuroimaging, particularly those that involve ataxia or coordination problems. Primarily, they are less likely to show neuroimaging abnormalities.

Q. I take it you reviewed the imaging

studies for Ms. Parker.

A. I reviewed the reports of the imaging studies. Before I would testify in a trial setting, I would insist on reviewing the films themselves.

Q. Is there anything -- and maybe what I'll do is I'll go over that with you, the imaging reports, but is there anything in the imaging studies that you recall now that seems to prove or suggest to you that this isn't ADEM?

A. Yes, there appeared to be what were described as multiple nodules of abnormal signal in the white matter of the centrum semiovale, and I think those findings are consistent with ADEM. There is a report -- I believe it was in December -- of an area of abnormality involving the frontal lobe that had some element of hemorrhage involved in that. That is somewhat atypical for ADEM.

There's a -- there's a related entity called acute hemorrhagic leukoencephalitis that's often considered a more severe form of ADEM in which case you see a small degree of hemorrhage. It's unclear to me whether or not that frontal lobe

abnormality seen in December is a manifestation of this more severe cousin of ADEM or not.

I don't think it's prudent to look at just imaging studies and, on imaging studies alone, say that this is ADEM or this is not ADEM. Radiologists typically say, this is consistent with the clinical picture of ADEM or it's inconsistent with it, but, like in all things, we consider the neuroimaging along with the laboratory evidence along with the clinical course to arrive at a clinical diagnosis.

Q. Have you ever diagnosed ADEM where you found no evidence of ADEM in the neuroimaging studies?

A. I don't have a recollection of personally having completely normal imaging, but that may be an issue of timing as well, but, no, I think in -- in my experience, most of the studies have been abnormal.

Q. And that's part of your analysis when doing your differential diagnosis, is to review and analyze neuroimaging studies to determine whether they are consistent with this diagnosis?

A. That's part of the picture, yes.

Q. In addition to the clinical picture that you also are reviewing in connection with making your diagnosis?
A. Right.
Q. Is there anything else that you reviewed in making the diagnosis of ADEM?
A. Other than the clinical course --
Q. Clinical and --
A. -- the laboratory studies and the neuroimaging?
Q. Right.
A. No, that pretty much encompasses my

practice of neurology.
Q. I'm going to again refer to Defendant's Exhibit 3 and ask you to -- I'll refer you to paragraph 6 of the Affidavit, which is your opinion.
A. Yes.
Q. And why don't you read that for the record and then tell me what the basis of your opinion is. I think you've said it, but let's go ahead and say that again.
A. Paragraph 6: Based upon my review of Luann Parker's records, I state that, within a

reasonable degree of medical and scientific certainty, her symptoms associated with ADEM (Acute disseminated encephalomyelitis) were caused by her vaccine received in October of 1998. Furthermore, additional symptoms associated with steroid treatment of ADEM represent secondary effects following the vaccination.

Q. Tell me the basis for that opinion.

A. As you said, we've already covered this in a sense. The basis for my opinion is that she had a clinical course that is consistent with ADEM, she had laboratory studies that were consistent, she had no --

Q. Can I stop you there?

A. Yes.

Q. Tell me what laboratory studies were consistent with ADEM.

A. We talked earlier about the spinal fluid studies that did not show evidence of an infectious encephalitis. We talked earlier about immunological studies that -- like sed rate -- that ruled out vasculitis, a whole host of other laboratory studies done in looking for vasculitis, looking for stroke. We discussed earlier the

normal angiography ruling out vasculitis as well. So many of the studies that are consistent with ADEM are consistent because they have successfully ruled out other -- other diagnoses.

We've also talked about the probability that ADEM occurs specific -- occurs following a specific trigger, a viral infection or vaccination, and we've established that an influenza vaccination was given two days before the onset of her symptoms. So, to the best of my recollection, that's the basis for my opinion.

Q. We haven't talked about the second part of your opinion concerning the additional symptoms associated with steroid treatment.

A. Yes.

Q. Can you explain what you mean by that?

A. It is not clear how best to treat ADEM; it's a difficult and challenging disorder. Because it has some similarities with multiple sclerosis, some physicians reasonably conclude that treating with high-dose steroids may be an effective way to mitigate some of the symptoms.

So Mrs. Parker's treating physicians made a decision to treat her with steroids because

they were concerned about her clinical condition. What they found was that she developed a new set of symptoms while on steroid therapy that basically had to do with confusion, disorientation, basically psychiatric-like symptoms.

Now, it's certainly the case that such symptoms may occur directly as the result of ADEM, although that's quite uncommon. It's also the case that such symptoms can occur as a consequence of steroid therapy, which her physicians felt was indicated in her case. So I was not prepared to conclude that her additional symptoms were directly referable to the ADEM but may have been caused by an intermediary of the requisite treatment.

Q. So I understand this, some of her

psychosis-like symptoms you're not saying were directly caused by ADEM but perhaps caused by the treatment that she received for the ADEM?

A. That's right. I don't have a way to convincingly distinguish between those two.

Q. Does ADEM, is that -- is it a consequence of ADEM that someone will develop a psychosis?

A. It can be, but it's uncommon.

(DFT. EXH. 4, FluzonePackage Insert, was marked for identification.)

BY MR. SICILIANO:

Q. Doctor, you have been provided with what has been marked as Defendant's Exhibit 4. Can you identify that for the record?

A. This is labeled, Influenza Virus Vaccine USP Trivalent Types A and B marketed under the name Fluzone, and it appears to be an FDA-approved package insert for the immunization.

Q. For the vaccine --

A. Yes.

Q. -- that was administered to Luann Parker?

A. I only note that it says 1998-1999 formula and that was the time when she received the

vaccine.

Q. You've indicated that you've prescribed or recommended influenza vaccine before?

A. That's correct.

Q. Have you ever read the package insert for influenza vaccine?

A. I'm sure, at one time, I have read it, yes.

Q. I just want to call your attention to the section on adverse reactions and specifically call your attention to a paragraph on page 6 of this package insert.

A. Yes.

Q. The fourth paragraph down, beginning with, neurological disorders.

A. Yes.

Q. Could you read that paragraph for the record, please?

A. Yes. It says, neurological disorders temporally associated with influenza vaccination such as encephalopathy optic neuritis, partial facial paralysis and brachial plexus neuropathy have been reported; however, no cause and effect has been established. Almost all persons affected were adults, and the described clinical reactions

began as soon as a few hours and as late as two weeks after vaccination. Full recovery was almost always reported.

Q. Doctor, do you consider that statement an accurate statement of the medical -- wait a minute, strike all that. Do you consider that statement contained in the package insert accurate?

A. As I indicated earlier, I've not reviewed the literature; specifically, I've not reviewed the two studies cited or footnoted with that statement. It may well be that there are an insufficient number of cases for epidemiologic study to establish a cause and effect relationship. I have no reason to disagree with that.

Q. Is the encephalitis that we are talking about, would that be encompassed in the description of encephalopathy in that paragraph?

A. Encephalopathy is a very broad term that basically refers to brain dysfunction of a variety of causes, infectious or metabolic, and I believe that it would encompass what we've specifically been talking about, ADEM.

Q. Do you intend to offer any opinions on -- as to the accuracy or the adequacy of the package insert marked as Defendant's Exhibit 4?

A. No, that's beyond my area of expertise.

MR. SICILIANO: Let's go off the record a second.

(Off-the-record conference.)

(DFT. EXH. 5, Test Results Summary dated 12/05/93, was marked for

identification.)

(DFT. EXH. 6, Verified Radiology Results dated 10/14/98, was marked for identification.)

(DFT. EXH. 7, Wellington Diagnostic Center Report dated October 14, 1998, was marked for identification.)

(DFT. EXH. 8, Radiology Report dated 10/22/98, was marked for identification.)

(DFT. EXH. 9, Radiology Report dated 10/23/98, was marked for identification.)

(DFT. EXH. 10, Radiology Report dated 10/28/98, was marked for identification.)

(DFT. EXH. 11, Riverhills Healthcare, Inc. Encounter Report dated 12/28/1998, was marked for identification.)

(DFT. EXH. 12, Wellington Diagnostic Center Report dated February 26, 1999, was marked for identification.)

(DFT. EXH. 13, Wellington Diagnostic Center Report dated June 25, 1999, was

marked for identification.)

BY MR. SICILIANO:

Q. Doctor, you've been handed what has been marked as Defendant's Exhibits 5, 6, 7, 8, 9, 10, 11, 12 and 13. And in general, can you tell me -- can you identify those records for the record?

A. These appear to be neuroimaging studies, both CT and MRI, for Mrs. Parker, the earliest being December 5th, 1993 and the last being June 25th, 1999.

Q. Let's start with Defendant's Exhibit 5. Just identify that one for the record.

A. All right, this is a CT head scan done in December 1993. My recollection is that this was done in the context of an evaluation for migraine headaches. The study was read as normal.

Q. And after reviewing it, do you have any --

(The proceedings were interrupted.)

BY MR. SICILIANO:

Q. Is it your impression that this is a normal CAT scan of her brain?

A. It has been read by the radiologist as

a normal study.

Q. Okay. I want you to look at Defendant's Exhibit 6 and tell me what that is.

A. The next study is also a CT scan done without contrast. The date is October 13th, 1998. That would be three days after she received her flu vaccination. This study is recorded as somewhat abnormal with mild dilatation of the cerebral sulci and cerebral ventricles.

What this is saying is that there is a little bit more space within and around the brain than one would expect for a patient of -- of this age perhaps. The radiologist considers this consistent with mild cerebral atrophy. We have no evidence that this study was compared with the one done five years previously.

Q. And I want to ask you about this study. The radiologist reading the CT study indicates that it is consistent with a mild cerebral atrophy. Tell us what that means.

A. When one speaks of atrophy, we're

talking about either a diffuse or a localized dropout of neurons so that there is not the abundance of brain cells that one would expect for

that age.

Q. What's -- what causes that?

A. There can be a variety of different causes. In older patients, we can see the development of moderate cerebral atrophy as a manifestation of a dementia, of Alzheimer's disease. In someone who has had a hypoxic ischemic insult, say after a cardiac arrest, we can see a moderate to severe atrophy as a consequence of previous insult to the neurons in the brain.

We don't have a great deal of information about whether this represents a significant integral change. Being able to compare the two CAT scans would be very helpful in that regard. Some radiologists are more sensitive in calling issues of atrophy. This may very well be the case, although, in my experience too, sometimes increased subarachnoid space or subarachnoid fluid or, put more simply, more space between the brain and the skull can be misinterpreted as brain atrophy.

Q. You don't know at this point?

A. Having not looked at the film, I can do nothing other than accept the report at face value.

What I can say is that none of the symptoms that Mrs. Parker was manifesting at that time or manifested over the next few weeks could be satisfactorily explained by the presence of cerebral atrophy alone.

Q. To put it more in lay terms, would cerebral atrophy be like loss of brain volume?

A. Yes.

Q. And at least this radiologist, looking at her CAT scan on October 13th, 1998, believed that she has less brain volume than a person of her age would normally expect to have?

A. That's correct.

Q. What kind of symptoms would you have with a loss of brain volume?

A. Probably the most sensitive symptom might be memory loss, short-term memory loss.

Q. Anything else?

A. That would -- that would be the initial one. I would not expect a patient with atrophy to present with the clinical history reported here of numbness and dizziness.

Q. The brain atrophy reported in this CT

scan, would that at all be related to receiving the

influenza vaccine?

A. Probably not, in that atrophy, as implied in this report, is -- is the reflection of an ongoing longer-term process. I would not expect atrophy to develop over three days.

Q. Let's look at Defendant's Exhibit 7 and tell me what that is.

A. Exhibit 7 is an MRI scan of the brain done with and without gadolinium. It's done --

Q. Okay, tell me what that means.

A. Let me just finish.

Q. Okay, go ahead.

A. -- it's done one day after the CT scan that we just discussed.

Q. So that's October 14th?

A. That's correct. Gadolinium is a paramagnetic contrast material that permits us to look at the integrity of the blood-brain barrier. In other words, can something get out of the bloodstream into the brain in violation of what is usually supposed to occur, which is a tight barrier between the two. The presence of enhancement with gadolinium tends to show us a breakdown of that blood-brain barrier, so it gives another look or

another dimension to our look at -- at brain function.

In this study, the radiologist notes first abnormal signal in the posterior parietal regions of the brain. Parietal area of the brain is that which interprets or regulates or integrates sensory phenomena, among other functions. And -- and while the clinical correlation here on the study is apraxia, it may be that these parietal findings explain symptoms reported elsewhere of numbness or tingling.

What is notable is that the greatest area of abnormality appears to be on the surface of the brain, on the covering of the brain, the meninges, as opposed to the deep white matter which we discussed previously as most characteristic of ADEM. However, the radiologist notes that, on the T2 weighted images, which tend to be more sensitive than T1 weighed images, he notes small areas of abnormal increased signal are noted in the adjacent brain parenchyma, primarily involving the cortex. That is the gray matter.

As -- as a footnote, while we talk about ADEM as being a disorder of the white matter, that's not exclusively the case. We know that

patients with ADEM can present with seizures, which is a phenomenon involving the cortex or the gray matter. So while ADEM focuses on demyelinating and is predominantly a white matter disorder, it's not exclusively so.

Q. Is there anything in this imaging study taken on October 14th, 1998 that leads one to the diagnosis of ADEM?

A. I would say, at this point, we have not seen characteristic changes of ADEM, that's correct.

Q. Talk to me a little bit about the meninges. What would be the cause of the changes in the meninges that are reflected in here?

A. Again, we're looking at abnormal enhancement, so we're talk -- probably talking about some sort of inflammation with some increased blood flow to that area. It could be, at this point, an infectious inflammation, or it could be a noninfectious autoimmune type inflammation. We don't have enough information in this MRI to distinguish between those two. And that's probably what would lead a clinician to do a spinal tap and make sure we're not dealing with a viral infection or bacterial infection.

Q. The radiologist in this case has set forth in his opinion -- or her opinion possible etiologies which include sarcoid; infection, either viral or inflammatory; ischemic changes, including vasculitis.

A. Right.

Q. Doesn't list ADEM?

A. However, item 3 under her opinion, which she doesn't give as much prominence to, does talk about this patchy abnormality in the white matter, which, at this point, is nonspecific but really heralds what we will be seeing later as the findings of ADEM. I think, in this particular study, the emphasis is on perhaps the more dramatic acute early inflammatory phase before some of the autoimmune part has really kicked in.

As -- as we'll discuss later, this meningeal enhancement is a transient phenomenon or phase, even as her clinical condition is worsening, and the clinical condition tends to be -- tends to track better with the increased abnormality in the white matter cells.

Q. Let's go to Defendant's Exhibit 8 and identify that for me.

A. Can we take a short break?

MR. SICILIANO: Absolutely.

(A recess transpired.)

THE WITNESS: Exhibit 8?

BY MR. SICILIANO:

Q. Yes.

A. Exhibit 8 is also an MRI scan with and without gadolinium. It was done six days after the previous MRI scan. What has appeared in the interval are multiple small nodular areas in the deep white matter of both parietal lobes. The radiologist indicates that most of these do not enhance.

Q. Tell me what that means.

A. What it means is that they don't light up with the gadolinium, suggesting that that is evidence of demyelinization causing the abnormal signal, but there is not yet evidence of breakdown of the blood-brain barrier allowing the gadolinium dye to cause them to light up.

As she correctly points out, these findings in and of themselves are nonspecific. They could represent a demyelinating disease, they could represent ischemia, they could represent infarction. The thing to note, however, they're distributed in a manner that's in -- unlikely to be

ischemic. First of all, they're punctate or regional or multiple, so that we're not seeing a ledge or a large area of ischemic brain that you would observe if a blood vessel were blocked, but this radiologist is not extending herself any further than just the images itself permit.

Now, she also notes that, higher in the parietal lobe, that there are some lesions that do enhance, indicating that they would represent more severe involvement where there was both demyelinization and breakdown of the blood-brain barrier. Good news is there's not significant swelling around these or mass effect that could cause secondary problems. She speculates they could be metastases, although that seems highly unlikely given the evolution of what we've seen in the clinical course.

She notes finally that there is still some of this enhancement of the meninges, which is a very nonspecific finding that in no way establishes or refutes the presence of ADEM.

Q. What's the cause of that?

A. It could be as simple as just increased blood flow to the meninges. It's sort of a

nonspecific inflammatory response. It does not

suggest a specific disease process, and it's physically remote from changes that we're seeing in the deep white matter of the brain.

It's not clear that that is a clinically significant finding in that we did not see it spreading to involve the meninges or the covering of the whole brain as we might with an encephalitis or a meningitis, and it just seems to be that particular region.

Q. But that's not consistent with the ADEM?

A. Nor is it inconsistent with the ADEM. It doesn't impact my impression one way or the other about that in the same way that mild cerebral atrophy doesn't affect my impression one way or the other.

Q. In looking at the October 22nd, 1998 study which is Defendant's Exhibit 8, is there more involvement of white matter than before?

A. There was an addendum dictated the following day where, in retrospect, she suggests that there was not a dramatic change over those between those two lesions.

MR. NAMEI: Is that exhibit 10 that she's --

BY MR. SICILIANO:

Q. That's exhibit 9, right; isn't that correct?

A. Right, that's correct, exhibit 9 is -- is the addendum. It's unclear whether or not she's adopted a different standard for interpretation or reporting, but she has made a conscientious effort to go back and review the two films and, in fact, when looking back, sees the same abnormalities in the earlier film that she has reported on the film six days later.

Q. So is the answer there really isn't a change from the prior film on the --

A. Well, she --

Q. -- as far as the involvement of the white matter?

A. -- she appears to conclude there has not yet been a dramatic change.

Q. She does say in Defendant's Exhibit 9, which is the addendum dated October 23rd, 1998, prime differential consideration is inflammatory processes involving the meninges in this area, as

meningitis which may be present on a viral or bacterial phase. Can you tell me what that means or how you interpret it?

A. Well, she's basically saying what we've already discussed, is that the cause of this meningeal enhancement is unclear; the differential diagnosis includes viral encephalitis, includes bacterial meningitis, includes those things that -- that need to be looked for. And she may not be aware that spinal fluid studies were in fact obtained on October 22nd and that, you know, there was only one white blood cell in the spinal fluid and that's well within the normal range.

So there seems to be some suggestion of an infectious process radiologically, but there is not confirmation of an infectious process when the physicians went back to look. So the radiologist is doing her job in terms of providing warning about things that the clinicians need to be looking for, but her suspicion and her concern does not establish the diagnosis.

Q. Let me go back to Defendant's Exhibit 8. The second paragraph of her opinion there says, there are, in addition, a few nodular and ovoid

enhancing lesions identified in the bilateral high parietal lobes. What are those, what's a lesion?

A. A lesion is a generic word for a spot or an abnormality. It really has no specificity in

terms of cause. What she's simply saying is that there are some nodular spots deep in the white matter close to the center of the brain, and, as you come closer to the surface of the brain, you not only have some more of these nodular spots, but you have some that she describes as ovoid, and some of those are now enhancing.

So the closer you get to the surface of the brain, the more these areas of abnormality include breakdown of the blood-brain barrier as well as demyelination.

Q. And what would be an explanation for that?

A. Could be different levels of severity of dysfunction within the lesion, it could represent a slight difference of timing of the appearance of the abnormalities, you know, where perhaps some of them are more advanced, and, therefore, there has been more breakdown of the blood-brain barrier. It doesn't necessarily imply

two entirely different causes of the abnormality.

Q. Are those specific findings consistent with ADEM?

A. Yes.

Q. Are they inconsistent with it at all?

A. I don't believe so. There is -- there is evidence of demyelinating lesions, and there is evidence of gadolinium enhancement. Both may be seen with ADEM. Now, I -- I have to state that I've not actually looked at the films, I'm reading between the lines of what looks like a thoughtful neuroradiologist.

Q. Are those findings typical of ADEM?

A. They're more typical of ADEM than they would be of multiple sclerosis, say, where the nodular demyelinating areas tend to be clustered more centrally around the ventricles. What we don't see is perhaps an area perhaps as large as one might expect with ADEM, although she doesn't really provide any specific information about the number or the size of these lesions.

Q. Let's go to Defendant's Exhibit 10 and tell me what that is.

A. 10 is yet another MRI scan done six

days after the last MRI scan. At this time, the patient's reported to have left arm weakness and difficulty walking. Now, in this particular report, the radiologist says that the ventricles and subarachnoid spaces are normal in appearance. That seems at some conflict with the CT scan of

October 13th where the ventricles and cerebral sulci were felt to be enlarged. There is concordance, however, in that a degree of atrophy in the sylvian fissure is -- is still present.

Q. And that's the loss of brain volume?

A. Right, sylvian fissure is the space between the temporal lobe and the frontal lobe. So that -- that is more generous, implying perhaps some atrophy of the temporal lobe. Again, multiple small nodular nonenhancing lesions are seen in the deep white matter.

Q. And it says there, not associated with acute mass effect. What does that mean or how would you interpret that?

A. Mass effect is secondary swelling and displacement of normal brain structures because of swelling. Classic example would be a brain tumor which may be relatively modest in size and yet have

surrounding it an area of edema or swelling or increased fluid within the brain. And that swelling can actually push brain structures out of the way and -- and cause symptoms secondary to displacement of brain structures.

Now, if we think back to the pathophysiology of ADEM, what we see is an

inflammatory -- I'm sorry, a demyelinating response around the veins deep in the substance of the brain. So if we're looking at a process that begins in the region of the veins and then spreads out around that, we would expect to see multiple small crops or areas of demyelinization representing the involvement of these multiple veins.

It doesn't make sense that, if that's your pathophysiology, that you would have any big lesion surrounded by a lot of edema that would cause mass effect. What the verbiage in the report is -- is probably more standard template, just saying, there's no mass effect, there's no displacement of immediate structures. One wouldn't expect that given what she's described is there.

She does note that, at this point,

there seems to be some diminution in the -- in the number of these area abnormalities -- areas of abnormality. The -- the more superficial ones closer to the surface of the brain don't appear to have diminished much at all, however. And at this point, the meningeal enhancement that's really been the focus of the attention on the last two MRI scans seems to be abating.

Q. Under her conclusions, she writes, no integral change in multiple small nodular areas of high signal intensity in the deep white matter of the deep parietal lobes without associated enhancement probably represents small areas of ischemia or infarction or could represent plaques related to a demyelinating process, stable in appearance on all MRIs compared. How do you interpret that?

A. Well, there's a little bit of inconsistency. In the body of the report, the radiologist says, there appears to have been some interval decrease and irregular areas of enhancement in the leptomeningeal region, so there is some improvement of the study. But you were talking about the de --

Q. Number 2.
A. -- demyelinating regions. I'm sorry, would you repeat your question?
Q. Number 2 under her conclusions, paragraph 2.
A. Right. Again, her differential diagnosis is reasonable; these are things that need to be considered. They do appear to be relatively stable at this point.

Q. So they're not getting worse?
A. They don't appear to be getting worse, and there's no areas of new enhancement, that's correct.
Q. In a typical ADEM, would you expect the white matter enhancement or lesions to be getting worse or better at this stage in time?
A. I would -- well, I would expect there to be a period of worsening and then a period of improvement. The period of improvement, however, can really be measured over weeks to months, it's not going to occur over days because, remember, there has been demyelinization, you know, the -- the myelin around the nerves has been destroyed, you know, it's got to be remanufactured and

repaired. You know, the blood-brain barrier has broken down; that's got to be repaired. It's -- that repair isn't going to occur over a matter of days.

Q. Well, what is typical for the progressive nature of ADEM? How long does it take normally?

A. I mean, usually the -- the symptoms evolve over a week or two or three, I mean, it's -- it's relatively acute, but again, it's important to

remember that radiologic abnormality is not always present with ADEM. And so, if you have a spectrum between no radiologic abnormalities and classic radiologic abnormalities, then you're going to have all radiant in between, and I think that, you know, that may -- that may best describe what we're seeing here.

Q. But typically the course of ADEM is you have a period, maybe days or a week or so or a couple of weeks, of worsening and then maybe a longer period of gradual improvement?

A. I will agree with that.

Q. And you --

MR. NAMEI: Just for clarification, are

you talking about symptoms or are you talking about the radiological manifestation, getting more sick?

MR. SICILIANO: Well, I'm actually asking about both.

MR. NAMEI: Because he answered it about radiological, but sometimes there's radiant in the spectrum that, you know, you can and you cannot see, whereas the symptoms getting worse and then they get better, it's like a bell curve.

BY MR. SICILIANO:

Q. Let's go to Defendant's Exhibit 11 and

identify that for the record.

A. Exhibit 11 is also an MRI. However, this one was done December 28th, so it's approximately two months after the last MRI scan. And this shows several changes. What we know is, over that two-month interval -- but we don't know when during that two-month interval -- there have been a couple of changes.

The first change is one of the enhancing areas noted previously now has some hemorrhage in it.

Q. Meaning bleeding?

A. That's right. So in -- we know that

first there was an area of demyelinization. Second, because this area enhanced, we know that there was a breakdown of the blood-brain barrier. And now we're seeing evidence of actual blood that has disbursed into this lesion, you know, itself having crossed the blood-brain barrier. So that is a new change.

What we don't have is information from the radiologist that permits us to date this blood. Neuroradiologists can often do that as the hemoglobin degenerates over time, and they can give you some sense of the duration of -- of the

hemorrhage, how long it's been there.

Q. Would it be typical that an ADEM would have this kind of hemorrhaging a couple of months now after the first onset?

A. Well, is it typical? No. But there are two points to be made. The first is that the study was done two months later. That doesn't necessarily tell us that the hemorrhaging occurred two months later. That's why I was talking about the importance of trying to date the hemorrhage.

The second, as I mentioned a while back, there is a variant of ADEM called this acute

hemorrhagic leukoencephalitis that is actually a more severe form of ADEM. In that particular disorder, this hemorrhagic transformation is seen more characteristically. So, no, this is not a common finding in ADEM but suggests the possibility of perhaps a more malignant course than we typically see with ADEM. Alternatively, it could represent some interplay between ADEM and her underlying conditions.

Q. Explain that.

A. Well, we know that the patient has hypertension, for example. It could be that, in this region of breakdown of the blood-brain

barrier, that a patient with hypertension is more likely to develop a secondary hemorrhage into that area of involvement than a patient that doesn't have hypertension. I mean, that's pure spec -- speculation on my part. I don't have enough information to -- to tell.

The second thing that is of concern is that there appears to be progression of the demyelinating process in the white matter. And there's -- there is sort of an irony here in that, while there appears to be more demyelinization,

there doesn't appear to be more enhancement. So there's not progression in the breakdown of the blood-brain barrier, but there has been evidence of demyelinization.

Now, one possible explanation for that could have been that the impairment of the blood-brain barrier has now resolved so that there's no more enhancement so that the lesion is no longer acute and what we're seeing two months later is the chronic appearance, you know, of what could have happened a month or six weeks previously. Again, I have no way to know that for certain.

Q. But is it typical -- again, we're

talking now December 28th, 1998 where there is a progression of the white matter disease -- is that typical in ADEM?

A. I would say that it is not typical; however, when faced with providing an alternative explanation or an alternative pathophysiology or an alternative disease that has yet to be diagnosed, described or discovered, it seems to be the most reasonable explanation, and given the congruence of clinical findings and radiological findings.

Q. If you were presented with this kind of finding now, more than two months after the initial onset, what would you be thinking about as far as an alternative diagnosis?

A. Well, first of all, as I mentioned, we need to be careful not to fall into the trap of assuming that these changes occurred two months afterwards, but with that -- but with that caution, we would be concerned about other demyelinating diseases. Does this represent a type of multiple sclerosis that we do know occurs at multiple points in time?

But again, the distinction is between a monophasic illness and an illness that has multiple events. What is unusual is that this is un --

fairly protracted, but I don't believe that it's with -- that it's outside of reasonable experience with ADEM because we do know that, in some cases of ADEM, the recovery phase, you know, extends for a year.

Q. Have you treated anyone that you diagnosed with ADEM that had a course consistent with this?

A. I have certainly had patients where, a

month into the process, they were getting worse when I was -- when I'd been telling the family they ought to be getting better, but I can't say that I have had a patient that had been exactly like this with the combination of the hemorrhagic transformation and the combination of the what appear to be late findings of -- of increased white matter signal.

But I -- but I don't believe that this pushes me beyond my comfort level in calling this ADEM, and I don't think it pushed her treating physicians beyond their comfort level in calling it that. It still represents the most feasible explanation for her clinical course.

Q. But if you were one of the treating physicians here, would you agree that these kinds

of findings would have caused you to be concerned that she does not have ADEM and that she may have something else?

A. Oh, they were very concerned. I mean, that's why they launched on an exhaustive workup to make sure there wasn't something else. You know, this was the timing of her really definitive evaluation where they looked at vasculitis, they

looked at risk factors for stroke, they looked at all of these other things that it could be other than ADEM, because there was some concern about timing.

And I think it's to their credit that they responded just as you suggest, that they go back and reconsider the original diagnosis, which -- which they very responsibly did.

Q. Let's look at Defendant's Exhibit 12 and tell me what that is.

A. Defense exhibit 12 is an MRI dated February 26th, 1999, so that is going to be two months after the last MRI scan. Again, I've not looked at the study, and it's somewhat more difficult to read between the lines, but several points are made. One is that there appears to be some areas of enhancement, again referring to

breakdown of the blood-brain barrier, perhaps in a slightly broader distribution than was seen before.

We also see evidence of new abnormality now seen in the thalamus, which is a deep gray matter structure in the center of the brain. The thalamus is what integrates all of the sensory input into the brain before relaying it off to the

cortex. Involvement of the thalamus is often seen in ADEM -- let me correct that: Is sometimes seen in ADEM. Often we see it earlier than we do in this case.

Another finding that's new are the possibility of petechial hemorrhages or pinpoint hemorrhages seen in regions of the brain. Now, it's unclear whether these are areas of hemorrhage that are superimposed or occurring in the areas of abnormal demyelination or if this is a separate process. I can't tell that from the report.

One thing we can -- are concerned with with petechial hemorrhages, it is possibly a hypertensive change in the brain, so if there had been a period of uncontrolled hypertension, that might account for the petechia.

Q. Would you again agree that this kind of finding now in February of -- end of February of

1999 isn't typical of the normal course of ADEM if she had it?

A. As we're continuing to see changes unfold this late, now four months after the initial event, it's occurring at a time where the changes may be less commonly seen with ADEM. The burden,

however, is still to offer an alternative explanation of an intercurrent illness or another process or an alternative process compared to what we typically see with ADEM.

Now, what we don't know is, has steroid therapy that she's received during the course of this treatment had some mitigating effect on the timing or the evolution of the process, is the immunological or autoimmune response that she has more sluggish or more delayed than with the average person for any number of reasons.

But the key question is whether there is anything that is substantively different that suggests an alternative diagnosis, and it's not clear to me that -- that we have evidence of an alternative diagnosis because we have just worked up, you know, in December, all of these alternatives and -- and found nothing.

Q. Let's look at Defendant's Exhibit 13.

Tell me what that is.

A. 13 is now four months after the last MRI or about eight months after the original studies, so now we see that there are no new areas of enhancement, we see that things are relatively

stable over the previous four months and that whatever process seemed to be active accounting for those February changes is now quiescent and -- and stable. So --

Q. It's not getting worse?

A. It's not getting worse.

Q. Is it getting better?

A. At this point, there's no evidence that these abnormal signals are going away or getting better; however, it's not unusual for there to be clinical improvement that antedates radiologic improvement by months. A patient with multiple sclerosis, for example, may have abnormal signal in the brain long after they have recovered from the attack of -- of MS.

So in -- in sum, what's atypical, if you will, about these series of studies is that the story plays out over a slightly longer period of time than we typically see with ADEM, but it still remains basically a monophasic illness with a

period of worsening followed by a period of stability and, I would presume, subsequently followed by a period of improvement.

Although I don't have current medical

records, my understanding is that she has clinically improved, and I would expect further neuroimaging studies to be the same or to show some mild improvement.

Q. You brought with you today a couple of textbooks. Tell me what those are. You've marked a couple of them too.

A. Right.

Q. Show me what you have marked too, but go ahead --

A. One is -- one is simply a neuroradiologist textbook, and the other is a general neurology textbook. What I marked were just the -- the citations that refer to ADEM. And, you know, I didn't know whether that would be relevant or helpful in today's deposition.

Q. Can we look at those? Let's just read the -- the names of the books.

A. All right, the first one is Diagnostic Neuroradiology by Anne Osborn. This is a very standard classic neuroradiology textbook. The

second one is one of two volumes of a neurologic comprehensive text called Diseases of the Nervous System, edited by Asbury, McKhann, McDonald,

Goadsby and McArthur.

Q. Let me look at the first one, please.

A. (Tendering)

(Off-the-record conference.)

BY MR. SICILIANO:

Q. Doctor, you brought with you today a couple of textbooks. Why don't you just identify the textbooks and the parts of textbooks that you have noted and tell me why you brought them with you today.

A. Not knowing whether it would be beneficial or not, I brought references that briefly cover the salient features of ADEM and the neurologic -- I'm sorry, in the neuroradiologic textbook, this spans pages 704 to 706.

Q. We just went over the neuroimaging studies of Ms. Parker. Does that textbook indicate that the neuroimaging studies that Ms. Parker has are typical of ADEM?

A. Well, the textbook demonstrates nodular subcortical lesions that may or may not enhance, as occurred in Mrs. Parker's case. The textbook also

demonstrates some abnormalities of the thalamus that Mrs. Parker developed later in her clinical

course. So, while I haven't actually seen her images, based upon the reports, there seems to be good concordance between the classic textbook description of ADEM and what is reported in her studies.

Q. Does the textbook at all indicate the influenza vaccine as a cause of ADEM?

A. The textbook, under the paragraph etiology, says, ADEM occurs in several settings, as follows... Item 3 of 4 says, following vaccination against rabies, diphtheria, smallpox, tetanus, typhoid or influenza.

Q. Okay, does it give any citation?

A. No, it does not.

Q. So you do not know what the basis for that kind of statement in that textbook is?

A. That's correct.

Q. Let's look at the second book you brought with you. And again, I would like a copy of the pages --

A. Sure.

(This page contains information to be supplied by counsel and/or the deponent.)

BY MR. SICILIANO:

Q. -- that you referred to.

A. The second reference is from a neurology text, and it discusses the clinical course of the disorder. And it spans pages 1673 to 1674, and it summarizes information that has appeared in the medical literature over the past half century.

Q. There's another section you have noted in this textbook.

A. There is. That basically talks about immune mechanisms and neurologic disease, the role of T cells. That is probably not as germane to our discussion.

Q. What pages is that?

A. What I have flagged is page 1512 to 1513. That focuses mostly on the mechanisms or the understanding to date of mechanisms of autoimmunity or immunological problems in the central nervous system.

Q. I understand you have reviewed the affidavits of two of the Defendants' experts in this case.

A. I believe three. I looked at affidavits from Mark Shilling, William Paul Glezen

and Richard Edward Latchaw.

Q. I'll not ask you about Mark Shilling's, but I will ask you about Dr. Glezen. Is there anything in his opinion that you take issue with?

A. Well, his opinion does not seem to address the clinical course of Mrs. Parker at all. He simply says that the package insert is accurate and provides adequate warning of potential adverse effects.

I'm not an expert on package inserts, but, you know, I will acknowledge that the insert provides warning that, you know, the vaccine should not be given to people with Guillain-Barre syndrome and should not be given to people who have active neurologic disease, presumably because there is evidence that it has an adverse effect on those conditions. I -- I really have no basis to -- to disagree with him.

Q. Okay. Let's look at Dr. Latchaw's opinion. Do you have any -- do you take issue with his opinion?

A. Dr. Latchaw's opinion is as brief as his CV is lengthy. I'm puzzled by Dr. Latchaw's opinion because, while a radiologist, he is offering judgment about clinical issues. And I

obviously take exception to it because his opinion is diametrically opposite to mine.

He provides no basis for his opinion, nor does he offer an alternative plausible explanation for Mrs. Parker's course. On the other hand, I wouldn't expect a radiologist to be able to offer a plausible alternative explanation for Mrs. Parker's course; that's what a clinical neurologist would do.

Q. One thing I didn't ask you about was that, at least according to Mrs. Parker, she had received influenza vaccine for a number of years prior to this one in 1998. Does that provide you with any indication of the cause and effect relationship between the influenza vaccine and her neurological course?

A. I don't know that that helps me one way or the other, knowing that each annual vaccine is slightly different from the other different viral antigen, different vehicle. While it intuitively may make sense that, if she's tolerated one, she would tolerate them all, you know, experience just doesn't seem to bear that out with vaccines in general.

MR. SICILIANO: Let me look at my

notes. I think we're done.

(Off-the-record conference.)

MR. SICILIANO: I don't have anything further. Thank you, Doctor.

(The deposition was concluded at 12:11 p.m.)

SIGNATURE OF DEPONENT

I, the undersigned, DAVID A. GRIESEMER, do hereby certify that I have read the foregoing deposition and find it to be a true and accurate transcription of my testimony, with the following corrections, if any:

PAGE	LINE	CHANGE	REASON

_____
DAVID A. GRIESEMER Date

CERTIFICATE OF REPORTER

I, Lisa F. Walkabout, Certified Shorthand Reporter and Notary Public for the State of South Carolina at Large, do hereby certify that the foregoing transcript is a true, accurate, and complete record.

I further certify that I am neither related to nor counsel for any party to the cause pending or interested in the events thereof.

Witness my hand, I have hereunto affixed my official seal this 4th day of August, 2003 at Charleston, Charleston County, South Carolina.

_____
Lisa F. Walkabout
Court Reporter
My Commission expires
November 14, 2004

I N D E X

REQUESTED INFORMATION INDEX

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